Perceptions of Asthma by Adolescents at Home*

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Objectives: **To test symptom perception in asthma under natural circumstances and to establish relationships between changes in airway obstruction as indicated by wheeze, dyspnea, general sensations, and emotional state.**
Design: **Continuous *in vivo* monitoring.**
Method: **Symptom perception was tested in 30 adolescents with severe, unstable asthma. They were continuously monitored in their homes for 72 h. Symptom perception was defined as the relation between self-reported dyspnea and airway obstruction as evident from audible wheeze. Tracheal sounds were continuously recorded with wireless telemetry for wheeze assessment. Dyspnea was assessed four times per day on a Likert-type 10-point scale, as well as four times randomly after pager remote command. The subjects kept records of use of medication, daily activities, general symptoms, and mood state in a diary.**
Results: **There were nine subjects with one or two wheeze episodes, another three subjects with three or four episodes, and one subject with almost continuous wheeze. The presence of wheeze in general related significantly to a rise (from individual baseline) in dyspnea of > 2.5 scale points. Acute wheeze was the best predictor of a rise in dyspnea, but prolonged wheeze correlated significantly with negative mood and general symptoms.**
Conclusion: **Patients with prolonged airway obstruction perceived symptoms less well and were more vulnerable to negative effects of asthma than patients with acute onset airway obstruction.**
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Key words: adolescents; asthma; dyspnea; symptom perception; wheeze

There is often no linear relationship between degrees of airway obstruction and self-reported dyspnea in asthma. Research has consistently shown that patients may report themselves symptom-free in the midst of an asthma attack, or conversely report dyspnea without airway obstruction.[1,2] The phenomenon of inaccurate symptom perception in asthma, first reported in the 1970s, is now widely considered a major threat for asthma management.[3–6] Nonetheless, bronchodilators are commonly prescribed, and many patients rely on them.[7] This optimistic position may, at first glance, be supported by studies that indeed have shown proper symptom perception in a majority of patients.[8,9] Impaired symptom perception would be restricted to patients with severe pathophysiology, impaired autonomic control of respiration, or a tendency for somatoform disorders.[10–12] However, symptom perception research is plagued by methodologic problems. Many studies leave concerns in this respect, which makes their conclusions dubious.

The first and most common method for studying symptom perception in asthma has been to test how closely dyspnea and lung function relate.[13] This simple design has often been combined with a challenge test. Dyspnea and lung function are repeatedly assessed during inhalation of doubling concentrations of agents that trigger airway obstruction. A first problem is the limited number of possible observations. Signal detection theory stresses that a hundred stimulus-response pairs are required for a proper assessment of perceptual capacity.[14] Moreover, a challenge test may be stressful to many patients, which naturally enhances their introspection and dyspnea magnitude.[15–17] On the other hand, some patients may feel confident in the medical setting and neglect their symptoms, reflected in absence or mild dyspnea during significant airway obstruction. So-called blunting to airway obstruction may actually be indifference to symptoms or neglect. The major concern about this research method is that patients are aware that airway obstruction is

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induced and that they selectively monitor for respiratory changes, which unnaturally influences their symptom perception.[18]

The second method for studying symptom perception refers to signal detection theory. Experiments have been conducted to remedy methodologic problems associated with the first method, as well as to omit ethical objections against inducing actual airway obstruction. The subjects breathe through a mouthpiece and respond to externally applied airflow interruptions by pressing a button.[19] Unfortunately, there are stringent physical, physiologic, and psychological differences between this method and actual airway obstruction, manifested in, for example, respiratory frequency, carbon dioxide level, perceived control, and negative emotions.[5,20,21]

The third method for studying symptom perception provides primarily positive conclusions about the perceptual capacity of patients with asthma.[8,9] They estimate their own peak expiratory flow rate, and subsequently measure this value with a mini peak flowmeter. The problem is that estimating a physical performance is completely different from experiencing dyspnea during an asthma attack.[8] There is no experimenter prompting accurate introspection during daily circumstances.[12]

Considering these methodologic concerns, and in the light of recent experimental research, it is doubtful that patients with asthma perceive airway obstruction accurately. Rather than a distinction in "good" vs "poor" perceivers, there are indications that probably all patients with asthma are vulnerable to inaccurate symptom perception under particular circumstances.[5,12]

A recent study on stress-induced asthma showed that adolescents with and without asthma were similarly frustrated, sad, or angry. However, only asthmatics experienced dyspnea, which could not be explained by changes in lung function or blood gases.[22] Probably, evoked activity in the sensory organs, specific or aspecific for airway obstruction, gives rise to perceptual processing, and activates stored memories to form the basis for the conscious experience of dyspnea.[23] This perceptual process is influenced by meaning, relationships, context, judgment, and past experience.[24,25] Consequently, seemingly relevant information contributes to dyspnea magnitude, irrespective of lung function or emotions. For example, adolescents in a physical exercise setting reported more dyspnea after false feedback of respiratory wheezing sounds, or false feedback of lung function values of 30% below the actual value.[26,27] General sensations may also enhance dyspnea in a situation which is associated with asthma, for example, nasal congestion or skin itching.[28]

In summary, patients with asthma are vulnerable to a biased symptom perception that limits the possibilities for laboratory research. *In vivo* studies in respiratory disease have been worthwhile in patients with hyperventilation syndrome,[29] nocturnal asthma symptoms,[30] and for spontaneous cough assessment.[31] *In vivo* studies have methodologic advantages over laboratory studies but have specific problems as well, for example, lack of fluctuations in target symptoms, large data sets, and patient compliance. In this light, the current study merely comprises preliminary results.

The validity and reliability of wheeze as a marker of airway obstruction has been confirmed.[32] The diagnostic sensitivity and specificity of any wheeze for a reduction in FEV_1 of $> 20\%$ after physical exercise was 86% and 99%, respectively.[33] Moreover, continuous sound recording has shown that the sensitivity of wheeze for a reduction in lung function increases over longer periods of sound recording. Airway obstruction may not be reflected in wheeze in each and every respiratory cycle as observed during laboratory studies.[33,34] However, continuous *in vivo* monitoring showed that wheeze is rarely absent in patients with airway obstruction.[35]

The aim of this study was to investigate dyspnea during spontaneous airway obstruction, the latter indicated by wheeze. An additional aim was to establish relationships between airway obstruction as indicated by wheeze, dyspnea, general sensations, and emotional state.

Materials and Methods

Subjects

There were 30 adolescents with asthma participating, 18 boys and 12 girls, aged 13 to 17 years (mean, 14.3; SD, 2.1). They enrolled via general physicians in Amsterdam and received equal payment for their participation. All subjects and their parents signed informed consent.

The severity of asthma was evaluated according to the guidelines of asthma management by the British Thoracic Society et al.[36] The subjects were using medication from "steps four and five," indicating severe asthma: oral prednisolone or large concentrations of inhaled corticosteroids. The subjects continued using medication during the monitoring period. Use of additional medication (particularly bronchodilators) was reported in a diary. After the subjects had been accepted for the study, they waited until dyspnea complaints occurred. They then called the research team and the monitoring period commenced in the forthcoming weekend.

General Procedure

The telemetry equipment was installed in the homes of the subjects. Usually, monitoring lasted from Friday afternoon until Monday morning. The subjects and occasionally one of the parents were trained in the proper handling of the telemetry equipment. This included changing batteries and audiocassettes

every 10 h and replacing the tracheal microphone after taking a shower. The subjects were instructed in the proper completion of the diary and trained/instructed in proper dyspnea assessment. The subjects received a pager for remote command to report dyspnea. Lung function and anxiety were measured at the beginning and before the end of the monitoring period. The audiocassettes with recorded tracheal sounds were analyzed at the Institute. Presence of wheeze was scored and related to dyspnea magnitude. All these analyses were restricted to the actual 72-h monitoring period.

Technical Procedures

Tracheal sounds were continuously recorded with a Continuous Respiratory Telemetry system (Emco Electronics; Assendelft, the Netherlands). The hardware consisted of a microphone, transmitter, receiver, and recording equipment. The electret microphone (range, 20 to 25,000 Hz within three decibels) was fixed in a polyester cover. It was placed over the suprasternal notch with a double-sided adhesive anti-allergic ring (ARBO T08; Tyco; Neustadt, Germany). The sounds were recorded by a Hitachi VT-F8290 (Hitachi; Tokyo, Japan) on the audio tracks of videocassettes with a capacity of ten hours. The transmitter and rechargeable battery were carried on a waistbelt. During recording at night, the transmitter and battery were kept in a bag placed beside the subject's pillow. The actual time of recording was indicated on the tapes on a parallel track, allowing sound intervals to be related in time to dyspnea assessments.

Measures

Assessment of Wheeze: Wheeze was defined as secondary sounds in the normal tracheal sound, varying from high-pitched wheeze to soft background buzzing or solitary rhonchi.[33–35] Wheezes were scored as absent or present.

The examiners listened to the tracheal sound periods that coincided with the approximately eight different dyspnea assessments available from each subject. The presence of wheeze during these intervals of 10 min preceding the dyspnea assessment was scored. In addition, the sound records were analyzed for signs of asthmatic reactions. To this end, random intervals of 30 s were analyzed for approximately each 15 min from nighttime recordings and 5 min from daytime recordings. Although nighttime recording was generally not included in the results of this study, these sounds were analyzed by one examiner only to assess asthmatic reactions during the monitoring period.

Wheeze episodes were divided into acute and prolonged wheeze. Acute wheeze commenced from a no-wheeze period of at least 6 h. In other words, acute wheeze indicated that subjects were "suddenly" confronted with airway obstruction.

Assessment of Dyspnea: Dyspnea was explained to the subjects as breathlessness, labored breathing, shortness of breath, or tightness of the chest. The degree of dyspnea was measured with a self-report Likert-type scale with 10 points. The responses ranged from 0 (no dyspnea) to 9 (very severe dyspnea). This simple scale has extensively been used in previous studies. For example, a mean reduction in FEV_1 of 29% after histamine inhalation in adolescents with asthma coincided with a rise in dyspnea of 4.2 scale points, and a mean reduction in FEV_1 of 30% after a free running task (standardized by means of heart rate) coincided with a rise in dyspnea of 2.5 scale points.[10]

The assessment of dyspnea was carried out four times a day, once before dressing in the morning, and the final one before going to sleep. The subjects were also instructed to report dyspnea on remote command four times per day. This instruction was a sound signal by a pager, randomly activated by the experimenter via the telephone.

An individual threshold value for dyspnea was used in the cross-tab analysis, consisting of a rise in dyspnea of ≥ 2.5 scale points. This baseline was recorded before the monitoring period when lung function revealed no deviation from the individual normal range and when there was no wheeze audible (see Data Analysis).

Assessment of Lung Function: The lung function was assessed immediately before and after the monitoring period with a spirometer (Spirosense; Tamaraco Systems, Lode BV; Groningen, The Netherlands). There were three trials, and the highest values were used for patient description. The FEV_1 and forced vital capacity were expressed as a percentage of the normal value predicted (see Table 1).

Assessment of Trait Anxiety: Trait anxiety was measured because it is known to influence subjective symptoms.[5] Trait anxiety was measured at the beginning of the monitoring period with a subscale of the Spielberger Inventory.[38] The scale consisted of 20 statements with responses varying from 1 (not at all) to 4 (very much). The total score ranged from 20 to 80 points.

Assessment of Negative Mood State (Diary): Mood state was measured by the end of each day with a short version of the "Profile of Mood States," consisting of 15 relevant items regarding negative mood, selected from the original subscales "tension/anxiety," "depression/dejection," and "anger/hostility."[39,40] The subjects were instructed to report their mood state across the day. The response options ranged from 0 (not at all) to 4 (very much). The total score was 0 to 60 points.

Assessment of General Symptoms (Diary): By the end of each monitoring day, the subjects completed an adjusted self-report version of the "Children's Behavior Checklist," subscale somatic complaints, comprising nine symptoms.[41,42] The responses ranged from 0 (not at all) to 4 (very much). The total score was 0 to 36 points.

Assessment of Used Medication and Activity (Diary): The subjects completed two questions with open answers about their extra use of bronchodilator or other medicines and type of exercise that day.

Data Analysis

The relationship between presence of wheeze in general with a rise in dyspnea of ≥ 2.5 scale points was tested in a cross-tab analysis with χ^2 and the ϕ association coefficient. The difference in dyspnea during acute wheeze and prolonged wheeze was *t* tested. The influence of a set of predictors on dyspnea was tested with a multiple regression analysis. The seven predictors were as follows: acute wheeze (dummy variable 0/1); prolonged wheeze (dummy variable 0/1); trait anxiety; mood; general symptoms; premonitoring lung function; and age. Dyspnea during acute vs prolonged wheeze was correlated with the former predictor variables by means of Spearman's rank correlation coefficient.

Results

There were nine subjects with one or two wheeze episodes, another three subjects with three or four

Table 1—*Lung Function and Trait Anxiety (n = 30)

Variables	Mean (SD)
Pretest FEV_1	89 (14)
Pretest VC	93 (12)
Posttest FEV_1	90 (12)
Posttest VC	89 (11)
Trait	29 (8.2)

*VC = vital capacity.

wheeze episodes, and one subject with almost continuous wheeze. None of the subjects needed emergency care during the monitoring period.

The 30 subjects completed 234 dyspnea assessments, of which 2 were not used in the analysis because they had been preceded by bronchodilator inhalation. The mean dyspnea was 1.8 (range, 0 to 6; SD, 1.7). The mean dyspnea during wheeze episodes (n = 13; 32 assessments) was 3.3 (range, 1 to 6; SD, 2.3). There were 16 dyspnea assessments associated with acute wheeze and 16 dyspnea assessments associated with prolonged wheeze. The mean dyspnea during acute wheeze was 3.8 (range, 2 to 6; SD, 2.2) and during prolonged wheeze 2.6 (range, 1 to 3; SD, 2.1).

The mood and symptom data from the diary were used in the statistical analysis. The mean daily item score on mood was 1.4 (SD, 1.1), and the mean daily item score on symptoms was 1.7 (SD, 1.2).

The cross-tab analysis showed a significant relation between presence of wheeze and a rise in dyspnea of ≥ 2.5 scale points from individual baseline ($\chi^2 = 12.50$, $\phi = 0.74$).

The difference in dyspnea between acute and prolonged wheeze was significant [$t(31) = 4.33$, $p = 0.034$]. This indicated that there was more dyspnea during acute wheeze.

The multiple regression analysis showed that 59% of the variance in dyspnea could be explained by a set of seven predictors (multiple $R = 0.78$; $p = 0.01$). Only three predictors were of individual significant influence on dyspnea (and one nearly so): acute wheeze ($\beta = 0.75$, $p < 0.001$); prolonged wheeze ($\beta = 0.46$, $p = 0.021$); mood ($\beta = 0.35$, $p = 0.040$); general symptoms ($\beta = 0.33$, $p = 0.052$).

There were no significant correlations between dyspnea during acute wheeze and other variables. The following correlations between dyspnea during prolonged wheeze and other variables were significant: mood (0.38, $p < 0.05$); general symptoms (0.39, $p < 0.05$).

Discussion

This study showed that spontaneous airway obstruction as indicated by wheeze in the homes of asthmatics related significantly to a dyspnea rise from baseline of ≥ 2.5 scale points. Acute wheeze predicted dyspnea better than did prolonged wheeze. However, only dyspnea during prolonged wheeze correlated significantly with negative mood and general symptoms.

These preliminary results suggested that adolescents with prolonged airway obstruction were more vulnerable to negative effects of asthma than adolescents with acute onset airway obstruction. The results suggested that patients with a tendency for prolonged wheeze did not increase their dyspnea magnitude and use of bronchodilators, which was reflected in a negative "set" of mood change and general symptoms.

The relatively poor perception of prolonged airway obstruction has previously been suggested by Turcotte and coworkers.[43] In a study with only eight subjects, they observed that late asthmatic reactions were less well perceived than acute reactions. Burdon and coworkers[44] confirmed that subjects who entered the laboratory with a diminished lung function were slightly less accurate in the perception of induced airway obstruction.

Habituation to prolonged symptoms could possibly explain blunted perception of prolonged airway obstruction. In a study with normal adults, dyspnea in some subjects decreased after prolonged external airflow obstruction, supporting the influence of habituation.[45] However, habituation and behavioral adaptation are difficult to distinguish. Subjects who diminish the metabolic demand of oxygen by keeping calm may experience less dyspnea than those pacing up and down in distress. Moreover, patients may be aware of their prolonged difficult breathing, but since no anxiety-provoking changes occur, they somehow get used to labored breathing, which is reflected in dyspnea magnitude. Rather than so-called blunting,[3] this would be neglect of symptoms or indifference to them. The analysis of the "physical activity" could have provided information about a diminished activity in patients during prolonged airway obstruction. Because the current design made it not possible for the subjects to leave their homes, the variance in activity was too small to allow proper conclusions. Continuous monitoring of subjects with asthma, including assessment of the number of meters walked or heart beat, could possibly better explain variance in dyspnea during wheeze episodes.[46]

The results of the study were negatively influenced by the limited number of asthmatic exacerbations. Although the monitoring period was intended to commence during exacerbations of asthma, only 13 of the 30 subjects actually manifested asthma. This was particularly remarkable because subjects and participants had been instructed to make an appointment for monitoring only when asthma symptoms were manifest. This once more subscribed to the introductory statement that patients may report dyspnea in the absence of airway obstruction. However, some subjects had to wait a few days before monitoring commenced and symptoms were gone.

Although the relationship between airway obstruction and dyspnea is generally considered an indication of the accuracy of symptom perception, this is not correct. What is important is that subjects perceive airway obstruction in order to take precautions, not the degree of dyspnea they experience. The awareness of a forthcoming attack of airway obstruction should instigate use of bronchodilator medication, irrespective of the magnitude of dyspnea. This study showed that merely acute changes are likely to trigger both use of medication and increases in dyspnea. Prolonged airway obstruction had neither effect, although general symptoms and negative mood state were enhanced. It is possible that differences in dyspnea were also related to the intensity of airway obstruction, with more dyspnea reported during severe obstruction. However, arguments against this lay assumption include the following: (1) previous research, (2) premonitoring lung function assessment in subjects with prolonged airway obstruction, and (3) the empirical observation that extreme airway obstruction is reflected in atypical sounds, rather than wheeze.[3,35]

Continuous *in vivo* monitoring of subjects with asthma seemed an excellent method of studying patients' ability to cope with asthma. However, the problem may be the proper and useful assessment of relevant variables, such as daily activities and emotional state. Moreover, compliance with a medical regime should not be tested with patients using corticosteroids, but with those with marked fluctuations in lung function, treated with "if needed" bronchodilators.

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